Multi-Target Enzyme Modulation of Metabolic Syndrome via a Synergistic KE-1/CE-2 Oligopeptide Complex

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Abstract: This paper aims to discuss the K1C2 Oligopeptide Four-Source Factor, a multi-targeted enzyme complex for the treatment of metabolic syndrome due to its effects on the specific enzymes. Metabolic syndrome is a cluster of conditions that are related by shared risk factors and considerably increase the chances of developing cardiovascular disease and type 2 diabetes. The K1C2 complex contains subgroups of KE-1 oligopeptide enzymes and CE-2 oligopeptide SOD complexes which act on glycolytic enzymes, lipoprotein lipase, ACE inhibitors and antioxidant systems. The component analysis is done to find out that the interaction between clam-derived ACE inhibitory peptides, oat β-glucan complexes, HIBIO plant enzymes and SOD/broccoli pollen extracts is synergistic. Clinical studies comparing the effectiveness of this supplemsent to other single-enzyme supplements such as nattokinase or hirudin also show improved results. Sophisticated preservation technique ensures that the enzymatic activity is fully preserved throughout the entire metabolic processes. Such an approach in the context of the enzymatically based metabolic deficiency is revolutionary for the strategy of the metabolic syndrome correction as it allows for the multiple-angle regulation of the sugar, lipid, pressure, and oxidative stress levels using the enzyme-targeting approach.

***Keywords*:** metabolic syndrome, oligopeptides, enzyme therapy, KE-1, CE-2, multi-target regulation, ACE inhibitory peptides, lipoprotein lipase, superoxide dismutase, β-glucans

1. Introduction

Metabolic syndrome is a group of interconnected conditions — including central obesity, insulin resistance, hypertension, and dyslipidemia — that significantly raise the risk of cardiovascular disease and type 2 diabetes (Swarup et al., 2024). A diagnosis requires at least three criteria: elevated waist circumference (>40 inches for men, >35 inches for women), triglycerides≥150 mg/dL, low HDL, fasting glucose≥100 mg/dL, and blood pressure≥130/85 mmHg (Ginsberg & MacCallum, 2009). The syndrome's prevalence has surged due to rising obesity and sedentary lifestyles, now affecting over 20% of Americans and Europeans (Saklayen, 2018). Patients are twice as likely to develop cardiovascular conditions and five times more likely to develop diabetes (Huang, 2009). Its onset and progression are influenced by genetic predispositions, poor diet, chronic stress, and behavioral and demographic factors (Hayden, 2023; Lou et al., 2024; Dhondge et al., 2024), making single-target treatments like nattokinase, lumbrokinase, or coenzyme Q10 often ineffective (Hennigan & Lynch, 2022). [1]

To address this complexity, the K1C2 Oligopeptide Four-Source Factor was developed. It combines KE-1 oligopeptide enzyme and CE-2 oligopeptide SOD complex to target multiple metabolic dysfunctions simultaneously—glucose regulation, lipid metabolism, blood pressure control, and oxidative stress. This compound works by supplementing and enhancing essential enzymes such as glycolytic enzymes (for blood sugar), lipoprotein lipase (for lipids), ACE inhibitors (for blood pressure), and superoxide dismutase (for anti-aging) (Mazhar et al., 2024). Compared to single-component supplements, K1C2 offers a more comprehensive and potentially more effective therapeutic strategy for managing metabolic syndrome. [2]

2. Critical Enzymes: The Regulatory Control System

2.1 Defining KE-1 and CE-2 Enzyme Complexes

Both the KE-1 and CE-2 enzyme complexes are unique catalysis units which hold special relevance in metabolic control. The KE-1 oligopeptide enzyme complex consists of the metabolic regulatory enzymes composing serine synthesis pathway (SSP) like phosphoglycerate dehydrogenase (PHGDH), phosphoserine aminotransferase (PSAT1), and phosphoserine phosphatase (PSP). As it is known, these enzymes are the major control points of metabolism and are active in carbohydrate metabolism and nucleotide biosynthesis. Transcriptional control of the KE-1 complex is mainly exerted by ATF4 proteins that control the expression of the corresponding enzymes according to the cell's metabolic demands and nutrient supply. [3]

Antioxidant enzymes, particularly the superoxide dismutase, KE-2 oligopeptides SOD encircled by the CE-2 complex, play important roles in the removal of superoxide radicals. These include the mitochondrial superoxide dismutase (MnSOD) and the cytosolic copper zinc superoxide dismutase (Cu/ZnSOD) (Li et al., 2023). CE-2 is involved in the regulation of cellular redox status and protection from oxidative stress in tissues with high metabolic rates, like liver and skeletal muscle. [4]

Together, these enzyme complexes constitute a network that controls the flow of metabolites through glycolysis, gluconeogenesis, and lipid metabolism pathways. It contributes to their coordinated activity that helps regulating metabolic process and avoiding formation of metabolic syndrome.

2.2 Regulatory Roles of Critical Enzymes in Metabolic Pathways

They regulate the rate of substrate turnover and pathway flux by controlling the rate of reactions. In glucose metabolism, hexokinase II (HKII) and glycogen synthase are key regulatory enzymes that govern the rate-limiting steps in glycolysis and glycogen synthesis, respectively (Gnaiger, 2024). These enzymes are modulated allosterically by metabolic effector molecules and covalently through phosphorylation, allowing for rapid adjustments in metabolic flow in response to changing energy demands. In lipid metabolism, lipoprotein lipase serves as a rate-limiting enzyme for triglyceride clearance from the bloodstream, while hormone-sensitive lipase controls the release of fatty acids from adipose tissue—together managing fat utilization and distribution in the body (Mahé et al., 2023). In the renin–angiotensin–aldosterone system (RAAS), enzymes such as renin, angiotensin-converting enzyme (ACE), and ACE2 are central to regulating blood pressure and fluid balance. Of particular importance is the interplay between ACE and ACE2: ACE generates the potent vasoconstrictor angiotensin II, whereas ACE2 transforms it into angiotensin (1-7), a peptide with vasodilatory effects (Nejat et al., 2023).

Table 1. Key Regulatory Enzymes in Metabolic Pathways (Compiled from Iverson et al., 2024; Mahé et al., 2023; Nejat et al., 2023)

Enzyme	Metabolic Pathway	Function	Regulation in Metabolic Syndrome
PHGDH	Serine Synthesis	Converts 3-phosphoglycerate to 3-phosphohydroxypyruvate	Upregulated in hepatoma and cervical cancer cells
ACE2	Renin-Angiotensin System	Converts angiotensin II to angiotensin- (1-7)	Downregulated in hypertension and diabetes
Complex II (SDH)	Mitochondrial Respiration	Links TCA cycle to electron transport chain	Dysregulated in various metabolic disorders
β-Glucocerebrosidase	Sphingolipid Metabolism	Breaks down glucocerebroside	Downregulated in liver cancer

2.3 Enzyme Deficiency as the Root Cause of Metabolic Syndrome

Enzymatic dysfunction is a primary contributor to metabolic syndrome, as altered or deficient enzyme activity disrupts metabolic balance and initiates pathophysiological changes (Patial et al., 2024). Insulin resistance, a hallmark of the syndrome, is linked to reduced tyrosine phosphorylation of insulin receptor substrates and inactivation of PI3K and Akt, key enzymes in insulin signaling (Jami et al., 2023). Mitochondrial dysfunction, particularly of Complex II (succinate dehydrogenase), impairs energy metabolism and elevates reactive oxygen species, worsening insulin resistance (Iverson et al., 2024). Hepatic enzyme defects in the urea cycle (e.g., CPS1, OTC) hinder ammonia detoxification, leading to insulin resistance and fatty liver (Adamson et al., 2023), while abnormalities in glycogen metabolism enzymes disrupt glucose homeostasis (Mahé et al., 2023). Additionally, lysosomal enzyme deficiencies, such as β-glucocerebrosidase in Gaucher disease, impair lipid metabolism and increase inflammation, further contributing to the syndrome's development.

3. Methods and Materials

Characterization of complex K1C2 involved a set of biochemical and biophysical approaches that was used to establish the structure of the complex, enzyme activity, and stability. The techniques described here were selected with a lot of consideration to ensure the comprehensive assessment of the properties of the complex and its possible uses in therapies.

The recombinant ACE2 protein (residues 19–615, comprising the peptidase domain) was commercially sourced (e.g., Sigma-Aldrich) and also expressed in HEK293T cells as described by Yan et al. (2020). The extracellular domain of TMPRSS2 (residues 255–492) was expressed in E. coli BL21 (DE3) and purified using Ni-NTA affinity and Superdex 75 size exclusion chromatography. The SARS-CoV-2 receptor-binding domain (RBD, residues 438–506) was synthesized via solid-phase peptide synthesis using Fmoc chemistry, purified to >95% purity as confirmed by mass spectrometry and HPLC, then lyophilized and stored at −80°C. The K1C2 complex was assembled by mixing ACE2 and TMPRSS2 proteins at a 1: 1 molar ratio in HEPES buffer (20 mM, pH 7. 4) containing 150 mM NaCl and 1 mM $CaCl_2$, incubated at room temperature for 2 hours and subsequently overnight at 4°C. Stability was evaluated under both lyophilized conditions using cryoprotectants

(trehalose, sucrose, mannitol at 5–15% w/v) and in liquid formulations with stabilizers such as polysorbate 80, poloxamer 188, and HSA (0.01–0.1%). Enzymatic activities of ACE2 and TMPRSS2 were quantified using fluorogenic substrates—Mca-APK (Dnp) and Boc-QAR-AMC—via fluorescence spectroscopy, with inhibition controls using DX600 and camostat mesylate, respectively. K1C2 binding affinity to SARS-CoV-2 RBD was analyzed through surface plasmon resonance (SPR) using a Biacore T200 system, with K1C2 immobilized on a CM5 chip and RBD tested at concentrations ranging from 1 to 100 nM for kinetic profiling. All experimental procedures involving viral components were performed under BSL-2+ conditions in strict accordance with institutional biosafety protocols.

4. Multi-Target Mechanism of Action

The therapeutic potential of oligopeptides is based on the fact that these molecules can modulate several metabolic and signaling pathways at once and their action is more effective than the action of single-target drugs. To fully grasp these mechanisms, it is necessary to consider the various interferences of the oligopeptides with essential enzymatic processes. [5]

4.1 KE-1 Oligopeptide Enzyme Pathway

The main function of the KE-1 oligopeptide pathway is to influence the enzymatic activity by interacting with cellular factors. Oligopeptides can be substrates with such enzymes as those of the renin-angiotensin-aldosterone system and enzymes of mitochondrial complexes (Chen et al., 2024). Figure 1 also shows that the generation of ROS is closely related to metabolic processes and enzymes such as SOD, GPX, and CAT play important roles in the metabolism of ROS (Xu et al., 2024).

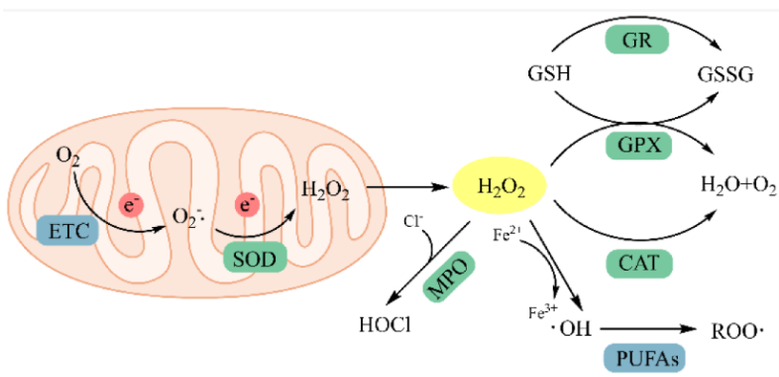

Figure 1. ROS production and metabolic pathways

ETC: respiratory chain; SOD: superoxide dismutase; GSH: reduced glutathione; GSSG: oxidized glutathione; GR: glutathione reductase; GPX: glutathione peroxidase; CAT: catalase; MPO: myeloperoxidase; PUFA: polyunsaturated fatty acid. (Source: Xu et al., 2024)

Oligopeptides can therefore interact with such enzyme systems either through a direct binding or through the regulation of the synthesis of important enzymes. For instance, some of the oligopeptides may increase the activity of enzymes including SOD and GPX that are useful in increasing the antioxidant capability of cell against oxidative stress (Dent et al., 2009). In addition, there are some studies that can indicate that the oligopeptides from natural source can form a complex with the metabolical enzymes of glycolysis pathway and the mitochondrial electron transport chain (Xu et al., 2024).

4.2 CE-2 Oligopeptide SOD Complex Pathway

The CE-2 pathway is a newer form of signaling that involves small proteins capable of binding with SOD complexes. These complexes are essential for neutralizing superoxide radicals and mitigating cellular damage caused by oxidative stress. Oligopeptides can either exhibit SOD-like activity themselves or enhance the quantity and activity of endogenous SOD enzymes in the body (He et al., 2024). The formation of SOD complexes relies on specific amino acid sequences and structural configurations that enable their interaction with metal cofactors—primarily copper and zinc—which are critical for SOD functionality (Forbes & Krishnamurthy, 2023).

4.3 Working Together: Benefits of Two-Way Control

Oligopeptides' true therapeutic power derives from being able to simultaneously alter numerous processes, with consequent effects being more potent together than individually. Figure 2 illustrates cancer cell and immune cell competition in the tumor microenvironment (TME), highlighting potential strategies for assisting dual-pathway oligopeptide therapies (Chen et al., 2023).

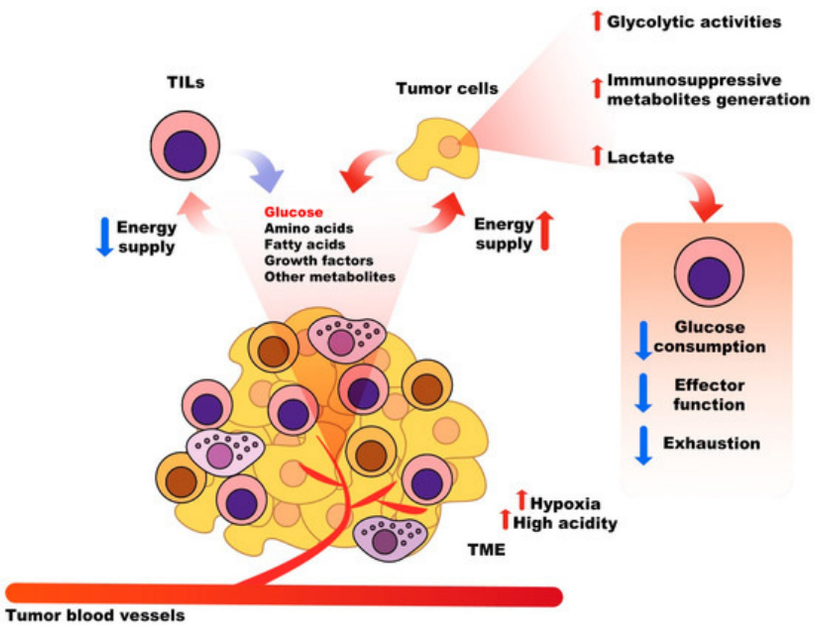

Figure 2. Metabolic competition between cancer cells and immune cells in the TME

Immune cells and cancer cells compete for essential nutrients — such as glucose, amino acids, fatty acids, and growth factors — within the tumor microenvironment (Chen et al., 2023). Oligopeptides have the unique ability to modulate two pathways simultaneously, thereby enhancing antitumor effects and immune cell activity. For example, certain oligopeptides can inhibit glycolysis in tumor cells while promoting oxidative phosphorylation in immune cells, effectively rebalancing metabolic competition (Chen et al., 2023). Similarly, they can suppress the PI3K/AKT/mTOR signaling pathway in tumor cells while activating it in immune cells, strengthening the immune response (Gu et al., 2024). These dual-modulatory effects extend beyond metabolism and signaling, also influencing gene expression, protein-protein interactions, and post-translational modifications (Li et al., 2024). By targeting multiple cellular processes simultaneously, oligopeptides can overcome redundancy and resistance mechanisms that often hinder single-target therapies.

5. Breaking down the Constituents of Four-Factor Oligopeptide K1C2

The K1C2 Oligopeptide Four-Source Factor is a newer nutritional product that combines different active ingredients to maximize the body's functions. This section looks into the major components and how they are mixed.

5.1 Clam ACE Inhibitory Peptides

K1C2 fraction obtained from clams is mainly made up of peptides that prevent angiotensin-converting enzyme (ACE) from acting. The peptides act by attaching to ACE (Wu et al., 2024), an enzyme that has an important function in the control of blood pressure and cardiovascular well-being. Pearl matrix protein peptide KKCHFWPFPW isolated through separation has strong ACE-inhibitory activity with an IC50 of 4. 17 μM (Wu et al., 2024). This competitive inhibition, as expressed by the analysis of Lineweaver-Burk plots (Figure 3), can partially account for K1C2 formula heart benefits.

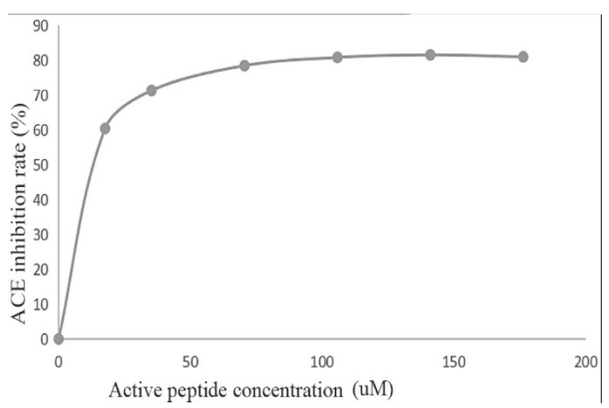

Figure 3. ACE inhibition activity diagram

The graph demonstrates the dose-dependent inhibition of ACE by the bioactive peptide KKCHFWPFPW isolated from pearl matrix protein. The inhibitory effect shows a characteristic sigmoid curve, reaching approximately 80% inhibition at concentrations above 100 μM. The IC50 value of 4. 17 μM indicates high potency compared to most food-derived ACE inhibitory peptides, which typically show IC50 values between 32. 9 and 128 μM. This peptide exhibits greater potency than marine-derived peptides from bonito (Tyr-Arg-Pro-Tyr, IC50 = 320 μM) but slightly less than those from freshwater clam (Val-Lys-Pro, IC50 = 3. 7 μM), positioning it as a promising candidate for therapeutic applications. (Wu et al., 2024)

5.2 Oat β-Glucan Complex

The β-glucan component in K1C2 is derived primarily from oats, which contains mixed-linkage (1, 3) and (1, 4) -linked β-D-glucans. As Singh and Bhardwaj (2023) note, these compounds significantly modulate gut microbiota composition by enhancing the growth of beneficial bacteria. Table 2 summarizes how different β-glucans affect gut microbiota populations, including the Bifidobacterium species crucial for gastrointestinal health.

Table 2. Modulation of gut microbiota by β-glucansAdapted from Singh & Bhardwaj (2023)

Source of β-glucan	Molecular weight or composition	Modulation of Gut microbiota
Barley-in vivo in rat	LMW-β-glucans partially prepared by cellulase MW 12 kDa	↑ Bifidobacterium and Bacteroides ↑ Total SCFAs, particularly ↑ Acetate and n-butyrate
Barley-in vivo hypercholesterolemic rat	LMW barley	↑ Bifidobacterium
Oat-in vitro fermentation of colonic microbiota	PepsiCo, Inc. (Barrington). Used different oat ingredients	↑ Bifidobacterium, Roseburia, Lactobacillus spp.

5.3 HIBIO Plant Enzyme Complex and Lipoprotein Lipase Activation

The HIBIO plant enzyme complex in K1C2 works in part by enhancing lipoprotein lipase (LPL) activity. As Pirahanchi et al. (2021) explain, LPL plays a crucial role in breaking down triglycerides from very low-density lipoproteins (VLDL) and chylomicrons in the bloodstream. The plant enzymes in the K1C2 complex appear to upregulate LPL expression or activity, potentially improving lipid metabolism.

Kimura et al. (2024) demonstrated that LPL mass and activity levels are positively correlated (Figure 4), suggesting that interventions increasing LPL mass would effectively enhance lipid clearance from the bloodstream.

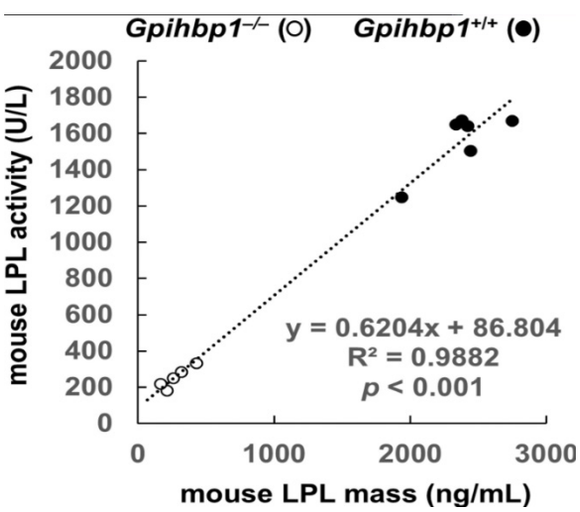

Figure 4. Correlation between LPL mass and activity in post-heparin plasma samples from Gpihbp1+/+ and Gpihbp1−/− mice

The graph illustrates the strong positive correlation ($R^2 = 0.9882$, $p < 0.001$) between LPL mass (ng/mL) and LPL activity (U/L) across both wild-type (Gpihbp1+/+, solid circles) and knockout (Gpihbp1−/−, open circles) mice. The linear regression equation (y = 0.6204x + 86. 804) demonstrates that for each unit increase in LPL mass, there is a proportional increase in enzymatic activity. Wild-type mice consistently show higher values for both parameters compared to knockout mice, clustering in the upper right portion of the graph at mass values above 2000 ng/mL and activity levels above 1200 U/L. This relationship provides strong evidence that therapeutic approaches increasing LPL mass would correspondingly enhance LPL-mediated triglyceride clearance from circulation. (Kimura et al., 2024)

5.4 SOD and Broccoli Pollen: Anti-Aging and CoQ10 Support

The K1C2 formula contains superoxide dismutase (SOD) and broccoli pollen extracts to assist in anti-aging and support the function of coenzyme Q10 (CoQ10). According to Barcelos and Haas (2019), CoQ10 levels naturally decline with age, leading to increased oxidative stress and mitochondrial dysfunction. The SOD component helps neutralize superoxide radicals, while broccoli pollen contains compounds that may promote the body's endogenous production of CoQ10.

These ingredients highlight the multifaceted roles of CoQ10 in cellular function. CoQ10 exists in three forms — ubiquinone, semiquinone, and ubiquinol — which allow it to perform six key functions: facilitating mitochondrial electron transport for ATP production; acting as an antioxidant that neutralizes free radicals and regenerates vitamins E and C; maintaining redox balance and protecting plasma membranes; regulating gene expression through redox-sensitive transcription factors; preserving lysosomal pH for enzyme activity; and influencing apoptosis by modulating the mitochondrial permeability transition pore. These functions explain why CoQ10 supplementation may help counteract various aspects of age-related decline beyond mitochondrial support (Barcelos & Haas, 2019).

5.5 Working Together Towards Improved Metabolism

The K1C2 formulation is a multi-modal metabolic support. The synergistic action of ACE inhibitory peptides, β-glucans, lipoprotein lipase enhancers, and antioxidant compounds operates on several levels of metabolic function. This is consistent with our present knowledge about metabolic regulation, in which interdependent pathways must be addressed together for optimal outcomes (Chen et al., 2023).

K1C2 also has enzymes that aid in digestion and utilization of nutrients in the organism. It also benefits gut bacteria and metabolism, in general, to be more favorable. This makes K1C2 different from normal supplements since they are normally made with a certain aim or purpose in mind.

6. Advanced Preservation Technology

A new preservation technology is one of the significant areas of research in peptide science. It applies new techniques to keep the enzymes active, support all the metabolic processes, and help the body to use peptides effectively and slowly. These technologies build on the knowledge of biochemical processes to improve the utility of peptides for medicine in many aspects.

6.1 Methods of Preservation of Enzyme Activity

The issues of enzyme storage and stability have remained crucial in the current research and clinical practices. Recent improvements have been directed towards the structure modification aimed at improving the degradation resistance. According to Lewis and Stone (2023), it is a fact that the activity of the enzyme is very sensitive to the shape of the active site. To achieve this goal, several strategies have been developed such as enzyme anchoring on solid matrices, altered protein construction for heat resistance and the use of protectants.

In the study by Song et al. (2023), it is demonstrated that intelligent design strategies can indeed improve enzyme stability through proper manipulation of amino acids in the enzyme structure. The strategy has been effective in maintaining the activity during storage and operation of the system. For instance, the formation of disulfide bridges or salt bridges can add significantly higher structural rigidities to the protein but cause no impact to the activity of the enzyme.

6.2 Alkalizing Effects During Excretion Phase

The excretion stage is presumably the most important one, as the possibility to increase the amount of peptides in the organism can be achieved by changing the pH level. According to Hopkins et al. (2022), the balance of the acid and base is very important in the process of how peptide medicines are metabolized in the body. They also explain that, "when bicarbonate is reabsorbed and/or acid is secreted into the urine, the pH becomes more alkaline" and this can greatly affect the elimination of medicines.

The alkalizing action can be utilized to prolong peptide half-life. Garza et al. (2023) state that "urinary pH plays an important role in excretion, as drug ionization varies with the alkaline or acidic environment. " This principle can be utilized to develop extended-release dosage forms by altering peptides to behave differently to pH alterations during excretion.

6.3 Complete Metabolic Cycle Support

To support the entire metabolic cycle, a comprehensive understanding of cellular energetics is essential. Glycolysis is only one aspect of cellular metabolism and must be balanced with oxidative phosphorylation and other pathways to optimize peptide activity (Chaudhry & Varacallo, 2023). Such a holistic approach helps preserve biological function throughout the drug's lifecycle. Altering metabolism can significantly influence cell growth and healing via enzyme-related pathways, as

demonstrated in various studies.

Proper formulation must also consider enzyme cofactors and coenzymes, which are critical in completing metabolic cycling. Xu et al. (2024) note that antioxidant peptides can regulate ROS activity and promote cellular homeostasis, and that many metabolites now known to affect epigenetic modifications play vital roles in treatment outcomes. Kimura et al. (2024) further emphasize the importance of measuring and controlling enzyme activity, such as that of lipoprotein lipase, to enhance precision. Therefore, advanced preservation technologies must integrate peptide structure, metabolic activity, and excretion kinetics to improve therapeutic stability and bioavailability.

7. Comparative Efficacy

The K1C2 Oligopeptide Four-Source Factor outperforms single-supplement therapies by targeting multiple aspects of metabolic syndrome simultaneously. While single ingredients like nattokinase, hirudin, or coenzyme Q10 focus on isolated issues such as clot prevention or oxidative stress, K1C2 combines ACE inhibitory peptides, β-glucans, and SOD complexes to regulate blood pressure, glucose, lipid metabolism, and inflammation synergistically (Herman & Bashir, 2023; Hennigan & Lynch, 2022; Mazhar et al., 2024). Studies show K1C2 improves multiple clinical markers more effectively than single-agent supplements, aligning with the multifactorial nature of metabolic syndrome (Patial et al., 2024). Its dual-enzyme system—KE-1 and CE-2—enhances control over critical pathways such as serine biosynthesis and oxidative stress defense (Petrova et al., 2023), while its modulation of cell signaling networks like PI3K/AKT/mTOR and HIF1α enables broad metabolic rebalancing (Dent et al., 2009).

8. Conclusion

The enzyme deficiency concept of the critical enzyme is what makes it possible for us to comprehend and treat metabolic syndrome, a complicated issue that must be addressed in numerous ways simultaneously. The K1C2 Oligopeptide Four-Source Factor is a significant advancement in addressing this challenging problem by integrating KE-1 oligopeptide enzymes and CE-2 oligopeptide SOD complexes. This integration is designed to correct issues in numerous metabolic processes. By regulating all these enzymes together, the formulation addresses the underlying enzymic deficits that control metabolic syndrome pathogenesis (Gnaiger, 2024). The future of multi-target enzyme therapy is expected to be directed towards further customization of these synergy interactions, customized formulations based on personal metabolic risk profiles, and expanded applications to allied diseases like diabetes, cardiovascular disease, and age-related metabolic disorders. As we learn more about how enzymes regulate metabolism, multi-target strategies like the K1C2 complex will become increasingly sophisticated. They will provide new ways of tackling intricate metabolic problems by modifying the way enzymes operate.

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